Amendments to the Specification:

Please replace the paragraph beginning at page 1, after the heading "FIELD OF THE INVENTION", with the following rewritten paragraph:

--The present application is a division of Serial Number 09/565,715, filed May 5, 2000, (hereby incorporated by reference herein) which is a continuation-in-part of Serial Number 09/444,327 that was filed on November 19, 1999 (hereby incorporated by reference herein) which claims priority from provisional patent application Serial No. 60/112,313, filed on December 14, 1998.--.

Please replace the paragraph beginning at page 1, after the heading "BACKGROUND OF THE INVENTION", with the following rewritten paragraph:

--Thrombin is an important serine protease in hemostasis and thrombosis. One of the key actions of thrombin is receptor activation. A functional human thrombin receptor (TR), cloned by Coughlin in 1991 (T.-K. Vu, *Cell* **1991**, *64*, 1057), was found to be a member of the G-protein coupled receptor (GPCR) superfamily. The receptor activation putatively occurs by N-terminal recognition and proteolytic cleavage at the Arg-41/Ser-42 peptide bond to reveal a truncated N-terminus. This new receptor sequence, which has an SFLLRN (Ser-Phe-Leu-Leu-Arg-Asn SEQ. ID. No. 1) N-terminus acting as a tethered ligand to recognize a site on the receptor, can trigger activation and signal transduction leading to platelet aggregation. Peptide analogues based on this hexapeptide have also shown good agonist activity leading to platelet aggregation. Since 1991, two other protease-activated receptors with extensive homology to the thrombin receptor, "PAR-2" and "PAR-3," were cloned, and found to be activated by similar N-terminal hexapeptide sequences. Hence, agonists / antagonists of the thrombin receptor, such as those included in this invention, may be useful in activating / antagonizing these protease-activated receptors as well.--

Please replace the paragraph beginning at page 30, after the headings "EXAMPLE 4", "*IN VITRO* THROMBIN RECEPTOR BINDING ASSAY," with the following rewritten paragraph:

--CHRF membranes (Jones, *Biochim. Biophys. Acta* **1992**, *1136*, 272) are thawed from -70°C, centrifuged at maximum speed for 5 min, washed twice with binding buffer (50 mM HEPES containing 5 mM $MgCl_2$ and 0.1% BSA), and re-suspended in binding buffer (25 µg/100 mL). 100 µl membranes are added to the 24-Wallac plates and delivered to the Tomtech apparatus. In a typical experiment, 6 µl of samples (from a 125 µg/mL intermediary plate, 20%DMSO) and 44 µl buffer are delivered to the plates (final conc. of compounds is 3.7 µg/mL, 0.6% DMSO). Similarly, 6 µl 20%DMSO and 44 µl buffer are delivered to both

column 1 (NSB) and column 12 (TB). 10 µl Ser-pFPhe-Har-Leu-Har-Lys-Tyr-NH_2, SEQ. ID. No. 2 (721-40; 500 µM in deionized water) is added to column 1. 50 µl tritiated 721-40 (specific activity 46 Ci/mmol) is added to all the wells. The plates are mixed well for 20 seconds, incubated for 30 min, and then harvested with 10 mM HEPES/138 mM NaCl using the Skatron harvester. The filters (GF/C Brandel FPXLR 296 filters are presoaked 3 h in 0.5% polyethylenimine in HEPES/0.1 M N-acetylglucosamine) are set in saran wrap and dried for 3 min in the microwave, and placed in sample bags (Wallac 1450-432). 4.5 mL scintillation fluid (Wallac, Betaplate Scint 1205-440) is added. The bags are sealed, placed in filter cassettes (Wallac 1450-104), and analyzed on the microbeta counter.--.